



Introduction

Akathisia directly translates to an inability to sit and is commonly described as internal restlessness. However, it can be difficult to describe by patients, leading to either misdiagnosis or underdiagnosis and can be a cause of medication nonadherence. Therefore. in this poster we will discuss this case in further detail, discuss the complex and nuanced nature of primary psychosis and medication side effects, and discuss the akathisia's suspected etiology, symptomology, and possible treatments.

Clinical Case

A 19yo Airmen was admitted involuntarily after presenting with disorganized thought process, perseveration on Airmen's creed with echolalia and auditory illusions (misinterpretation of intercom system), dyssomnia, and hyperreligiousity. The patient required several rounds of emergent medications during the initial days of admission. The patient was started on Olanzapine 10mg with improvement in organized thought processing and overall functioning. A trial of Ziprazidone was ineffective is continuing stability requiring re-starting Olanzapine. Olanzapine was titrated to 20mg. The patient had Parkinsonian side effects requiring Benztropine, with effectiveness noted a 1mg BID. During the second trial of Olanzapine, the patient began to experience increased paranoia and increased psychomotor activity. The patient would pace the unit (or rock/walk in place), walk up to staff asking about the safety of her family. Initially trialed on BID dosing of Propranolol. Once initiation, a remarkable and dramatic decrease in paranoia and psychomotor activity was appreciated. Propranolol was adjusted to the long-acting formulation and titrated to 120mg daily.

Akathisia or Psychosis: A case of how difficult determining side effect vs. primary psychosis can be

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Pathophysiology of Akathisia^{1,2,3,7}

Unclear and poorly understood

Primary theorized mechanism - related to low dopaminergic tone

- -imbalance of DA and 5HT/NE system
- -DA neurons in Ventral Tegmental Area and Substantia Nigra
- Hypothesized another neurotransmitters involved
- -GABA, glutamate

Medications associated^{1,2,3,4,5,7,8,10,11}

Antipsychotics – most notable

- -High potency, High doses, rapid escalation of med -> increased Akathisia
- -multiple trials show higher rates in FGA
- --Haloperidol specifically
- -For SGA
- --Aripiprazole and Lurasidone have higher rates

SSRIs, Anti-emetics, MAOIs, TCAs, Calcium Channel Blockers, Illicit Drug Use (Stimulants)

-have been linked to akathisia

Symptoms 1,2,3,5,7,9

Diagnostic Tool

- -Barnes Akathisia Rating Scale Most Widely Used
- -no widely accepted consensus

Dependent on the internal interpretation of inner restlessness

- -Intense dysphoria
- -irritability
- -agitation
- -anxiety
- -paranoia
- -Increased suicidality
- -Increased motor activity (pacing, repetitive movements)

Treatment^{1,2,3,7} Akathisia Alter Antipsychotic Regimen Add "Anti-Akathisa" Agent Reduce dose Switch to SGA First Line Switch to Lowpotency FGA Blocks NE/5HT inputs into DA pathways BID or TID (40-120mg daily) Others Mirtazapine 5HT_{2a} receptor antagonist 15mg daily 5HT₂₂ antagonists Benzodiazepines Cyproheptadine (8-16mg GABA - short term daily) - Anticholinergic SE Lorazepam (1-2mg) Mianserin (15mg/day) Clonazepam (0.5-1mg) Diazepam (5-15mg) Trazodone (100mg) -Anticholinergics 2a/c antagonism Ach/DA in nigrostriatal system Benztropine (1.5-8mg daily) Biperiden (2-6mg daily) Clonidine Trihexyphenidyl (2-10mg daily)

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Department of Defense or its Components

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